

YSTEM:OS - DIALOG OneSearch
 File 155:MEDLINE(R) 1951-2005/Aug W1
 (c) format only 2005 Dialog
 File 55:Biosis Previews(R) 1993-2005/Aug W1
 (c) 2005 BIOSIS
 File 34:SciSearch(R) Cited Ref Sci 1990-2005/Jul W5
 (c) 2005 Inst for Sci Info
 File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
 (c) 1998 Inst for Sci Info
 File 340:CLAIMS(R)/US Patent 1950-05/Aug 09
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Set	Items	Description
? s bhl or bh2		
	412	BH1
	875	BH2
S1	1036	BH1 OR BH2
? s bad		
S2	37363	BAD
? s s1 and s2		
	1036	S1
	37363	S2
S3	46	S1 AND S2
? s bcl?		
S4	82651	BCL?
? s s3 and s4		
	46	S3
	82651	S4
S5	46	S3 AND S4
? s heterodimer??		
S6	44102	HETERODIMER??
? s s5 and s6		
	46	S5
	44102	S6
S7	4	S5 AND S6

? rd

>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.

...completed examining records

S8 3 RD (unique items)

? t s8/3,k,ab/1-3

8/3,K,AB/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

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10840203 PMID: 7834748

Bad , a heterodimeric partner for Bcl -XL and Bcl -2, displaces Bax and promotes cell death.

Yang E; Zha J; Jockel J; Boise L H; Thompson C B; Korsmeyer S J

Howard Hughes Medical Institute, Department of Medicine, Washington University School of Medicine, St. Louis, Missouri 63110.

Cell (UNITED STATES) Jan 27 1995, 80 (2) p285-91, ISSN 0092-8674

Journal Code: 0413066

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To extend the mammalian cell death pathway, we screened for further **Bcl -2** interacting proteins. Both yeast two-hybrid screening and lambda expression cloning identified a novel interacting protein, **Bad**, whose homology to **Bcl -2** is limited to the **BH1** and **BH2** domains. **Bad** selectively dimerized with **Bcl -xL** as well as **Bcl -2**, but not with **Bax**, **Bcl -xs**, **Mcl-1**, **A1**, or itself. **Bad** binds more strongly to **Bcl -xL** than **Bcl -2** in mammalian cells, and it reversed the death repressor activity of **Bcl -xL**, but not that of **Bcl -2**. When **Bad** dimerized with **Bcl -xL**, **Bax** was displaced and apoptosis was restored. When approximately half of **Bax** was heterodimerized, death was inhibited. The susceptibility of a cell to a death signal is determined by these competing dimerizations in which levels of **Bad** influence the effectiveness of **Bcl -2** versus **Bcl -xL** in repressing death.

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...Descriptors: physiology--PH; *Carrier Proteins--metabolism--ME; *Proto-Oncogene Proteins--metabolism--ME; *Proto-Oncogene Proteins c- **bcl -2**

Gene Symbol: **bad**

Chemical Name: Antibodies; **Bad** protein; Carrier Proteins; Macromolecular Substances; Proto-Oncogene Proteins; Proto-Oncogene Proteins c- **bcl -2**; Recombinant Proteins; **bcl -x** protein

8/3,K,AB/2 (Item 1 from file: 55)

DIALOG(R)File 55:Biosis Previews(R)

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0009667922 BIOSIS NO.: 199598135755

Bad, a Heterodimeric Partner for Bcl -X-L and Bcl -2, Displaces Bax and Promotes Cell Death

AUTHOR: Yang Elizabeth (Reprint); Zha Jiping; Jockel Jennifer; Boise Lawrence H; Thompson Craig B; Korsmeyer Stanley J

AUTHOR ADDRESS: Howard Hughes Med. Inst., Div. Mol. Oncol., Dep. Med., Washington University Sch. Med., St. Louis, MO 63110, USA**USA

JOURNAL: Cell 80 (2): p285-291 1995 1995

ISSN: 0092-8674

DOCUMENT TYPE: Article; Literature Review

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: To extend the mammalian cell death pathway, we screened for further **Bcl -2** interacting proteins. Both yeast two-hybrid screening and lambda expression cloning identified a novel interacting protein, **Bad**,

whose homology to **Bcl -2** is limited to the **BH1** and **BH2** domains. **Bad** selectively dimerized with **Bcl -x-L** as well as **Bcl -2**, but not with **Bax**, **Bcl -x-s**, **Mcl-1**, **A1**, or itself. **Bad** binds more strongly to **Bcl -x-L** than **Bcl -2** in mammalian cells, and it reversed the death repressor activity of **Bcl -x-L**, but not that of **Bcl -2**. When **Bad** dimerized with **Bcl -x-L**, **Bax** was displaced and apoptosis was restored. When approximately half of **Bax** was heterodimerized, death was inhibited. The susceptibility of a cell to a death signal is determined by these competing dimerizations in which levels of **Bad** influence the effectiveness of **Bcl -2** versus **Bcl -x-L** in repressing death.

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8/3,K,AB/3 (Item 1 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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06445160 Genuine Article#: YT745 Number of References: 31

Title: Dimerization properties of human BAD - Identification of a BH-3 domain and analysis of its binding to mutant BCL -2 and BCL -X-L proteins (ABSTRACT AVAILABLE)

Author(s): Otilie S; Diaz JL; Horne W; Chang J; Wang Y; Wilson G; Chang S; Weeks S; Fritz LC; Oltersdorf T (REPRINT)

Corporate Source: IDUN PHARMACEUT INC,11085 N TORREY PINES RD/LA JOLLA//CA/92037 (REPRINT); IDUN PHARMACEUT INC,/LA JOLLA//CA/92037

Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1997, V272, N49 (DEC 5), P 30866-30872

ISSN: 0021-9258 **Publication date:** 19971205

Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814

Language: English **Document Type:** ARTICLE

Abstract: **Bad** , an inducer of programmed cell death, was recently isolated from a mouse cDNA library by its ability to bind to the anti-apoptotic protein **BCL -2**. Sequence analysis suggested that **Bad** was a member of the **BCL -2** gene family that encodes both inducers and inhibitors of programmed cell death. To further analyze the role of **BAD** in the network of homo- and heterodimers formed by the **BCL -2** family, we have cloned the human homologue of **BAD** and assessed its biological activity and its interactions with wild type and mutant **BCL -2** family proteins. Our results indicate that the human **BAD** protein, like its mouse homologue, is able to induce apoptosis when transfected into

mammalian cells. Furthermore, in yeast two-hybrid assays as well as quantitative in vitro interaction assays, human **Bad** interacted with **BCL -2** and **BCL -X-L**. Sequence alignments of human **BAD** revealed the presence of a BH-3 homology domain as seen in other **BCL -2** family proteins, Peptides derived from this domain were able to completely inhibit the dimerization of **BAD** with **BCL -X-L**. Thus, as previously shown for **BAX**, **BAK**, **BCL -2**, and **BCL -X-L**, the BH3 domain of **BAD** is required for its dimerization with other **BCL -2** family proteins. **BAD** was further analyzed for its ability to bind to various mutants of **BCL -2** and **BCL -X-L** that have lost the ability to bind **BAX** and **BAK**, some of which retain biological activity and some of which do not. Surprisingly, all of the mutated **BCL -2** and **BCL -X-L** proteins analyzed strongly interacted with human **BAD**. Our data thus indicate that mutations in **BCL -2** and **BCL -X-L** can differentially affect the **heterodimeric** binding of different death-promoting proteins and have implications concerning the relationship between heterodimerization and biological activity.

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...Identifiers--PROGRAMMED CELL-DEATH; HOMOLOG BAK; APOPTOSIS; INHIBITION; **BCL -X(L)**; GENE; DISTINCT; **BH1**

? log off

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10aug05 09:17:30 User231882 Session D1461.2
$1.00      0.295 DialUnits File155
$0.21      1 Type(s) in Format 4 (UDF)
$0.21      1 Types
$1.21      Estimated cost File155
$1.90      0.322 DialUnits File55
$2.00      1 Type(s) in Format 4 (UDF)
$2.00      1 Types
```

\$3.90 Estimated cost File55
\$6.84 0.309 DialUnits File34
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\$13.27 Estimated cost File34
\$2.08 0.094 DialUnits File434
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\$3.05 0.175 DialUnits File340
\$3.05 Estimated cost File340
OneSearch, 5 files, 1.195 DialUnits FileOS
\$1.06 TELNET
\$24.57 Estimated cost this search
\$24.62 Estimated total session cost 1.406 DialUnits

Logoff: level 05.06.01 D 09:17:30

You are now logged off